Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	46	Schmidt NEAR ann	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/01/11 15:08
L2	7	tumor ADJ invasion ADJ assay	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/01/11 15:08
L3	546	(cell ADJ migration ADJ assay) and (tumor cancer)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/01/11 15:17
L4	176	Ruoslahti NEAR Erkki	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/01/11 15:10
L5	3	I4 and (membrane NEAR invasion)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/01/11 15:13
L6	20	I4 and (tumor NEAR invasion)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/01/11 15:13
L8	435	I3 and (tumor WITH inhibit\$4)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/01/11 15:17
L9	11998	(invasion migration) SAME (tumor cancer)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/01/11 15:19
L10	1148	(invasion migration) SAME (tumor cancer) SAME (amphoterin cadherin integrin hyaluronic)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/01/11 15:20
L11	501	(invasion migration) WITH (tumor cancer) WITH (amphoterin cadherin integrin hyaluronic)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/01/11 15:22
L12	273	(invasion migration) WITH (tumor cancer) WITH (integrin)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/01/11 15:22
L13	4	(invasion migration) WITH (tumor cancer) WITH (integrin).clm.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2005/01/11 15:22

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(FILE 'HOME' ENTERED AT 15:24:43 ON 11 JAN 2005)

	FILE 'MEDLINE, CAPI	LUS' ENTERED AT 15:24:57 ON 11 JAN 2005
L1	2307303 S TUMOR	OR NEOPLAS? OR TUMOUR OR CANCER
L2	2275423 S INVAS	OR MIGRATION OR GROWTH
L3	282445 S L1 (L	L2
L4	107661 S L3 AN	O INHIBIT?
L5	3429 S L4 AN	O (INTEGRIN OR AMPHOTERIN OR CADHERIN OR HYALURONIC)
L6	937 S L5 AN) PY<=1998
L7	197 S L6 AN	O ASSAY
L8	149 S L7 AN	O INTEGRIN
L9	149 FOCUS L	3 1-
L10	98 S L9 AN	O (A(L)INTEGRIN)

- L11 ANSWER 10 OF 98 CAPLUS COPYRIGHT 2005 ACS on STN
- AN 1997:137748 CAPLUS
- DN 126:155848
- TI Attachment, spreading and migration of melanoma cells on vitronectin. The role of .alpha.v β 3 and .alpha.v β 5 integrins
- SO Experimental Dermatology (1996), 5(6), 308-315 CODEN: EXDEEY; ISSN: 0906-6705
- AU Van Leeuwen, Robert L.; Yoshinaga, Iara G.; Akasaka, Toshihide; Dekker, Sybren K.; Vermeer, Bert Jan; Byers, H. Randolph
- Recent in situ studies suggest the .alpha.vβ3 integrin is a tumor progression marker in melanoma. We analyzed 5 human melanoma cell lines for their expression of the vitronectin binding .alpha.vβ3 and .alpha $.v\beta$ 5 integrins using flow cytometry. The role of these receptors in cell attachment, spreading and migration was investigated using attachment assays, video time lapse spreading and migration assays and with function blocking monoclonal antibodies. Cell lines derived from later stages of tumor progression exhibited high levels of .alpha .vβ3 expression, whereas no similar correlation with . alpha .vβ5 expression was identified. Cell attachment, spreading and migration response on vitronectin correlated well with the expression level of the .alpha.v β 3 but not the . alpha. $v\beta5$ vitronectin receptor. Blocking of the . alpha.vβ3 integrin resulted in a significant decrease in cell attachment, spreading and motility whereas the function blocking antibody against the .alpha.v\beta5 integrin only inhibited cell attachment in cell lines with the highest level of expression of this integrin. Taken together, our study indicates that the level of expression of the .alpha.vß3 and .alpha.vβ5 integrins is heterogeneous in melanoma cell lines and that the .alpha.vβ5 integrin, if present, may function only during the initial cell attachment whereas the <code>.alpha.v</code> β 3 plays an important role in cell spreading and cell migration as well.

- L11 ANSWER 5 OF 98 CAPLUS COPYRIGHT 2005 ACS on STN
- AN 1994:677755 CAPLUS
- DN 121:277755
- TI A novel in vitro assay system for transendothelial tumor cell invasion: Significance of E-selectin and .alpha.3 integrin in the transendothelial invasion by HT1080 fibrosarcoma cells
- SO Clinical & Experimental Metastasis (1994), 12(4), 305-14 CODEN: CEXMD2; ISSN: 0262-0898
- AU Okada, Tomoko; Okuno, Hiroaki; Mitsui, Youji
- AB The interaction of tumor cells with endothelial cells is a key event in tumor metastasis. The authors established an in vitro invasion assay system, in which the invasion of tumor cells after interaction with endothelial cells can be examined Two chamber culture wells separated by porous membrane were used. Human umbilical vein endothelial cells (HUVEC) were placed on porous membranes coated with matrix components. The invasion by HT1080 fibrosarcoma cells was determined in this system by counting the number of cells that moved through the membranes from upper to lower chambers. HUVEC cells did not migrate through the membranes as judged by the staining with UEA-I. Observation by SEM revealed that HT1080 cells bound to HUVEC surfaces and migrated underneath the HUVEC monolayer. Effects of antibodies specific for cell surface adhesion mols. on the migration of HT1080 cells were examined Invasion of uncoated membranes and membranes coated with HUVEC cells was compared. Antibody against E-selectin significantly suppressed an increase of HT1080 cell invasion of HUVEC monolayers stimulated by IL-1 β or TNF.alpha.. Antibody against integrin .alpha .3 subunit remarkably inhibited the invasion of HUVEC cell-coated membranes, suggesting that integrins with the . alpha.3 subunit may play an important role in the transendothelial invasion by HT1080 cells.

- L11 ANSWER 4 OF 98 CAPLUS COPYRIGHT 2005 ACS on STN
- AN 1990:588996 CAPLUS
- DN 113:188996
- TI Monoclonal antibody and synthetic peptide inhibitors of human tumor cell migration
- SO Cancer Research (1990), 50(15), 4485-96 CODEN: CNREA8; ISSN: 0008-5472
- AU Yamada, Kenneth M.; Kennedy, Dorothy W.; Yamada, Susan S.; Gralnick, Harvey; Chen, Wen Tien; Akiyama, Steven K.
- The processes of migration and invasion by human tumor cells are likely to involve specific cell surface receptors, such as receptors for the extracellular matrix mols. fibronectin, laminin, and collagen. This study examined the roles of several of these receptors using a set of monoclonal antibodies directed against the $\beta1$ integrin family, as well as a series of synthetic peptides reported to inhibit various interactions of each of these proteins with the cell surface. The most general inhibitor of tumor cell migration was found to be the anti- β 1 monoclonal antibody 13, which inhibited the migration of human HT-1080 fibrosarcoma cells, 5637 bladder carcinoma cells, VA13 viral transformants, and HCT 116 colon carcinoma cells when fibronectin was the migration substrate. Moreover, this antibody was particularly effective in blocking cell migration on laminin, as well as migration within 3-dimensional collagen gels. It also inhibited in vitro invasiveness in a reconstituted basement membrane invasion assay (Matrigel assay) at concns. as low as 1 μ g/mL. Integrins of the β 1 class thus appear to play a central role in several types of migration by a variety of human tumor cell lines. Anti-.alpha.5 fibronectin receptor monoclonal antibody 16 also significantly inhibited migration on fibronectin, but not on other substrates, in 3 of the 4 cell lines. Conversely, anti-. alpha.2 monoclonal antibody F17 strikingly inhibited migration in 3-dimensional collagen gels, but not on other substrates, implicating the .alpha.2β1 integrin system in migration of tumor cells within collagenous matrixes. A series of synthetic peptides previously reported to inhibit interactions of normal cells with fibronectin, laminin, and collagen were also tested as inhibitors of tumor cell migration. Peptides containing the Arg-Gly-Asp adhesive recognition signal were partially inhibitory, but with occasional exceptions, most other peptides had no effects on migration. The results indicate the central importance of several specific $\beta 1$ integrins in human tumor cell migration and show the effectiveness of monoclonal antibody treatment in blocking this process in vitro.